ond noncontiguous fracture exists. Screening evaluation of the entire axis from occiput to sacrum is necessary and should be done rapidly so that the backboard can be removed to prevent skin decubiti. Identified fractures require further delineation with computed tomography or magnetic resonance imaging. Once mechanical stability has been assessed, treatment of the spinal axis can be planned. Treatment decisions include restoring stability, preventing delayed deformity, immobilization versus surgery, and the timing of surgery.

There is currently no cure for the neurological deficits incurred with spinal cord injury. Medical management of the patient focuses on avoiding secondary injury and rapidly instituting methylprednisolone therapy as appropriate. Adequate resuscitation of the patient begins the process of restoring homeostasis. Hypotension and hypoxia must be avoided scrupulously and treated aggressively if they do occur. Some experimental models suggest that maintenance of cord perfusion pressure is beneficial. This is accomplished by adequate intravascular volume repletion and judicious use of pharmacological pressors. Treatment of accompanying systemic injuries should be carefully coordinated. A multidisciplinary approach facilitates injury management and transition to rehabilitation thereby attempting to minimize the emotional impact upon the patient.

Methylprednisolone in high dosage has shown sufficient sustained benefit in SCI to merit routine usage and has been the standard of care in the US since 1990. An initial bolus of 30 mg/kg over 15 minutes is followed by a 23-hour continuous infusion at 5.4 mg/kg per hour. Timing of drug administration is crucial; the bolus must be initiated within 8 hours of injury. New evidence has just been published from a double-blind prospective randomized trial which studyied tirilazad, a new aminosteroid with potent antioxidant effects, versus 48-hour methylprednisolone therapy as opposed to the traditional 24-hour methylprednisolone therapy. The group treated with solumedrol for 48 hours experienced significant improvements in motor function at 6 weeks and 6 months, particularly the group who received the initial bolus within 3 to 8 hours of injury. Those who received the initial bolus within 3 hours of injury showed similar recovery patterns with all three treatment regimens. Patients treated with tirilazad showed improvements intermediate to the two steroid patterns.

In the optimal management of spinal cord injury, high-dose methylprednisolone therapy is indicated. If the initial bolus is started within 3 hours of injury, 24-hour treatment is appropriate; if the bolus is not administered until 3–8 hours, treatment should be extended for a total of 48 hours. The therapy should not be given more than 8 hours after injury.

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Management of Internal Carotid Artery Occlusion

ACUTE OCCLUSION of the internal carotid artery produces a variety of clinical syndromes, including complete absence of symptoms, fluctuating neurological deficits, or various degrees of completed stroke. In the general population, the incidence of asymptomatic internal carotid artery occlusion in people older than 60 years has been estimated to be approximately 1%. Conversely, 27% of patients with occlusion documented by angiography were found to have a history of neurological symptoms, including transient ischemic attack (TIA) and stroke. In prospective studies, the reported annual rate of ipsilateral stroke in patients with asymptomatic internal carotid artery occlusion has been estimated at 4–5%, two thirds occurring ipsilateral to the occluded artery.

Strokes and TIAs after internal carotid artery occlusion have been attributed to four main pathogenic mechanisms: 1) emboli into the circle of Willis from the distal end of the occlusive thrombus; 2) emboli originating from a residual internal carotid artery "stump" through the external carotid artery; 3) emboli from ipsilateral common carotid artery, external carotid artery, or aorta through collateral channels; or 4) hypoperfusion distal to the occlusion. Because of the capacity of the cerebral circulation to develop collateral flow, chronic hypoperfusion after internal carotid artery occlusion is relatively uncommon. In this condition, autoregulation is impaired and cerebral blood flow is susceptible to changes in blood pressure or cardiac output. This concept led to the widespread application of extracranial-to-intracranial bypass procedures in the 1970s. Although a randomized, prospective trial showed no benefit from this bypass for preventing stroke in patients with internal carotid artery or middle cerebral artery occlusion, there may be a subset of such patients with truly impaired cerebral autoregulation who would benefit from surgical revascularization to augment flow. To identify this subgroup, cerebral vasodilating stimuli such as CO_2 or acetazolamide can be used to dilate the vascular bed transiently and determine the adequacy of cerebral hemodynamic reserve by transcranial Doppler monitoring or single-photon emission computed tomography (SPECT) imaging.

Distinguishing between high-grade internal carotid artery stenosis and chronic occlusion is critical, since the two conditions are treated in different ways. Whereas carotid endarterectomy has been shown to reduce stroke

risk significantly in patients with symptomatic and possibly asymptomatic carotid stenosis, reconstructive surgery on chronically occluded carotid arteries is generally not attempted. Even when patency can be re-established, there is a high rate of re-occlusion, with catastrophic consequences. Noninvasive diagnostic modalities such as carotid artery ultrasound and magnetic resonance angiography have limitations for the diagnosis of carotid occlusion. Although advances in non-invasive imaging of the carotid artery will likely reduce or eliminate the need for conventional angiography, the clinician must be aware of the limitations of these tests before determining treatment.

In patients with acute carotid occlusion and profound neurological deficits, rapid emergent endarterectomy has been reported to yield good results occasionally. In one study, collateral flow on the preoperative angiogram was a good prognostic factor, and an associated middle cerebral artery embolus was a poor prognostic factor. Other studies showed no outcome difference between patients with restored patency of the internal carotid artery and those in whom patency was not restored. The duration of occlusion, extent of reduction of flow, degree of collateral filling, and tissue vulnerability are critical variables that contribute to the size of a focal ischemic area and its penumbra. Therefore, if surgery is to pursued, it should be done as rapidly as possible, since the duration of ischemia is one of the most critical factors influencing outcome. Many restrict emergent endarterectomy for carotid occlusion to patients with documented occlusion (e.g., hospital inpatients post-angiography) who do not respond to intra-arterial thrombolysis.

Both prospective and retrospective data have highlighted the increased risk of ipsilateral stroke during carotid endarterectomy in patients with contralateral carotid occlusion. In carotid endarterectomy trials for symptomatic and asymptomatic stenosis, contralateral occlusion imposed both a higher perioperative stroke rate and a higher risk of stroke in the nonsurgical patients. In patients undergoing carotid endarterectomy contralateral to carotid occlusion, a higher percentage of intraoperative electroencephalographic and blood flow changes occurs, requiring the use of an intraluminal shunt to maintain cerebral blood flow.

In summary, the diagnosis, evaluation, and treatment of patients with carotid artery occlusion is complex and based largely on anecdotal and retrospective experience. Although dramatic recoveries have been reported after restoration of blood flow in an acutely occluded artery, reconstruction of a chronically occluded internal carotid artery is probably of little value. Occasionally, patients with internal carotid artery occlusion and hemodynamic cerebral ischemia may benefit from extracranial-to-intracranial bypass procedures.

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Traumatic Brain Injury

TRAUMATIC BRAIN INJURY remains a major international public health problem responsible for hundreds of thousands of deaths annually. In addition to its mortal consequences, its morbidity affects millions of people, a minority of whom are damaged for life. In addition to the primary injury that occurs at the moment of impact, many macrostructural and neurochemical events occur from that time on. Clearly, prevention is imperative and is the best mechanism to limit injury. The success of seat belts and, more recently in the US, airbags is testimony to the cost-effectiveness of such preventive measures. Nevertheless, many still suffer major injuries that result in the initiation of secondary processes. Much of the focus in recent years has been on the consequences of hypotension and hypoxia and their prevention or early treatment.

One consequence of both the primary impact injury and the secondary insults is the development of brain swelling resulting in increased intracranial pressure (ICP). There has been considerable controversy about the mechanism by which ICP rises in the absence of an intracranial hematoma. For the last two decades, it was generally accepted that most of the increase in intracranial volume was caused by vascular engorgement (an increase in intravascular volume). New data suggest that in the majority of patients, at least during the first several hours and probably during the first several days, the major component responsible for increases in intracranial volume and pressure is cytotoxic edema.

These data were generated using a new magnetic resonance imaging technique called diffusion weighted imaging, which identifies the random translational motion of the protons of water, allowing the tracking of their location within the brain. A reduction in the apparent diffusion coefficient (ADC) indicates that water is moving into the cell, i.e., cytotoxic edema.

The observations with regard to edema are in keeping with magnetic resonance imaging studies using gadolinium that have failed to demonstrate the movement of contrast into the brain during the first 72 hours after head injury. This indicates that vasogenic edema, a mechanism previously thought to be responsible for water accumulation in the brain, plays little or no role within the first few days. While cytotoxic edema appears to be the predominant mechanism in some patients, increases in intravascular volume do occur.

As a result of these recent reports, new drug therapy can be aimed at brain edema. Moreover, the availability